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CASE REPORT

A Rare Clinical Presentation of Aortic Dissection

Ching-Chih Liu[1], Shaojung Li[2], Ko-Chiang Hsu[3]

[1]Departments of Emergency Medicine and [2]Cardiovascular Surgery, Taipei Medical University, Wan Fang Hospital, [3]Department of Emergency Medicine, Taiwan Adventist Hospital, Taipei, Taiwan, Republic of China

Aortic dissection (AoD) is one of the most common catastrophes involving the aorta. Nevertheless, early diagnosis remains to be a challenge in the emergency department (ED), particularly in young individuals. The diagnosis of acute AoD is often delayed secondarily to its propensity to masquerade as other illnesses (i.e., renal colic, spinal-cord injury, and acute cauda equina syndrome) that result in acute lower-back pain (LBP). Here, we report a 38-year-old man who presented to our ED because of acute LBP with one leg numbness and weakness. It was subsequently found to have acute Type A AoD. This young man was diagnosed early in the ED and treated promptly without major adverse sequelae. We highlight that ED physicians should always keep high alertness in vascular emergencies (i.e., acute AoD), whilst engaging a patient with an acute-onset severe LBP with one leg numbness and weakness.

Key words: Lower-back pain, aortic dissection, limb cynosis

INTRODUCTION

Acute lower-back pain (LBP) is one of the most common complaints for people to come to the emergency department (ED). Up to 84% patients have experienced acute LBP at least once in their life time.[1] Some of them are life-threatening due to its broad differential diagnosis and frequent disassociation between intensity of the symptoms, signs and seriousness of underlying pathology. The diagnosis of acute aortic dissection (AoD) is often delayed secondarily to its propensity to masquerade as other illnesses (i.e., renal colic, spinal-cord injury, acute cauda equina syndrome) that result in acute LBP. Here we present a 38-year-old male who presented to our ED with initial neurologic symptoms mimicking a herniated intervertebral disc (HIVD) of the lumbar spine with a significant neurological deficit, and finally diagnosed of acute Type A AoD.

CASE REPORT

A 38-year-old male was brought to our ED because of acute onset LBP developed 1 h before his admission. He also complained of left lower leg numbness and weakness. There was no chest pain or referring to back pain in his mention in present illness. He denied to have any traumatic history recently and knowledge of either genetic disease (i.e., Marfan syndrome) or lifting heavy weight objects lately. He also reported a significant history of hypertension and renal insufficiency. The remainder of the past medical history and review of systems were unremarkable.

On arrival, his vital signs were stable, apart from a blood pressure of 212/124 mmHg. On physical examination, we detected no typical characteristics of marfanoid appearance or connective-tissue disease and no audible heart murmur. The muscle power was normal 5/5 in 4 limbs with normal deep tendon reflex. The straight leg raising test is also normal in 70° bilateral legs. However, we disclosed an evidential skin color difference between his lower legs and feet. The left lower leg is pallor than the right one [Figure 1]. In addition, the pulsation of the left dorsalis pedis was diminished to palpation.

The blood pressure of four limbs was recorded as follows: The right arm 212/124 mmHg, the left arm 197/127 mmHg, the right lower leg 186/118 mmHg, but no blood pressure was measured on the left lower leg. Because the peripheral pulses and skin color were unequal between lower extremities, vascular insufficiency of the left lower leg was suspected. Contrast-enhanced computed tomography (CECT) of the chest to pelvis was arranged for the impression of AoD. A CECT revealed Stanford Type A AoD [arrow, Figure 2a]. Enhanced CECT scan showed true lumen compression by enlarged false lumen [arrow, Figure 2b], thus diminishing the pulsation [arrow, Figure 2a]. A cardiovascular surgeon was consulted immediately at ED and Bental procedure was

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Corresponding Author: Dr. Ko-Chiang Hsu, Department of Emergency Medicine, Taiwan Adventist Hospital, No. 424, Sec. 2, Bade Road, Taipei 105, Taiwan, Republic of China. Tel: +886-2-2771-8181 ext. 2756; Fax: +886-2-2741-8359. E-mail: kc0611@gmail.com

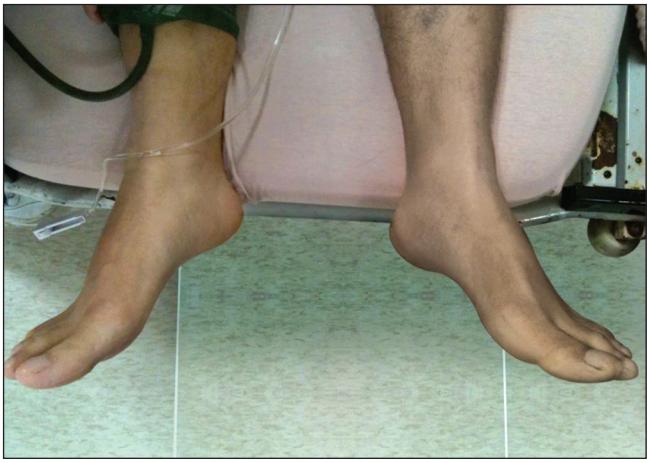

Figure 1. Skin color difference between legs

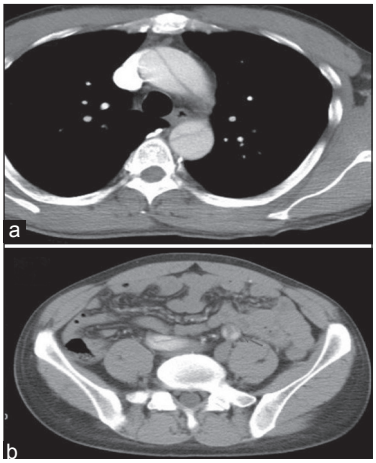

Figure 2. (a) Enhanced contrast-enhanced computed tomography (CECT) scans shows the minimal flap from ascending aorta through descending aorta. (b) Enhanced CECT scans showed true lumen compression by enlarged false lumen. Intimal flap was noted in bi-iliac artery

arranged, re-implantation of coronary artery into the graft. He was discharge under stable condition after 4 weeks hospital course. On follow-up at our patient department, his was free from LBP and his blood pressure was around at 120/80 mmHg under adequate medication control.

DISCUSSION

Aortic dissection is one of the most common catastrophes involving the aorta. It requires a timely diagnosis and if the condition is unrecognized and untreated, the mortality is nearby. Here, we report a young man who presented to the ED with acute LBP associating with left lower leg weakness and numbness, mimicking HIVD. It was found to have Type A AoD by a timely CECT. In a review of 464 patients from the International Registry of Acute Aortic Dissection (IRAD), the mean age was 63 years.[2,3] In the IRAD analysis, hypertension plays an important factor in young patients (<40 year), the result showed 34% had a history of hypertension in the young group. When comparing with older patients (>40 year), young patient have other unique risk factor that contribute to AoD, such as Marfan syndrome, bicuspid aortic valves.[2] For the present case, the patient had no evidence of Marfan's syndrome, sarcoidosis, defective collagen metabolism, or vasculitis. The only contributing factor would be hypertension. However, an apparently healthy individual presenting with acute unexplained out-of-proportional LBP with significantly skin color difference, indicating ischemic change, could be the "red-flag sign" of acute AoD.

The difficulty of diagnosis in acute AoD increasing when patient comes to ED without specific complaint such as chest pain radiating to the back. Especially, when younger patient arriving at ED without mentioning specific predisposing factors, whist an AoD is more easily to be overlooked. In our case, the patient had no history of connective-tissue disease and only about left leg numbness with LBP, it is easily to be impressed as a sciatica. Although chest pain is the most common complaint (nearly 100%[2]), but some literatures also report "painless" AoD, even up to 6.3%.[4] Painless dissection patient has more prior history of diabetes, aortic aneurysm, or cardiovascular surgery. In one study, they found out up to 10% patient could present neurologic symptoms, but without chest pain, which is compatible with our patient.[5]

In the IRAD analysis, the pulse deficit is more in Type A dissection than in Type B dissection (19–30% vs. 9–21%),[2,6] which have higher mortality rate and complication than those without pulse deficit.[7] Our patient not only presented with pulse deficit, but also an obvious skin color change. The skin color difference is an important clinical clue to suspecting a vascular insufficiency, which is mainly due to ischemic change, resulting from intimal flap or compression by intramural hematoma.

Moreover, the classic suggestive signs in chest radiography of AoD have been reported to be present in up to 50% of cases, nevertheless, only 33% of young patients had such findings,[8] which was normal in our case. CECT has a great advantage in diagnosis of acute AoD, sensitivity ranging from 83% to 98% and the specificity ranging from 87% to 100%.[9,10] Therefore, prompt CECT should be arranged if clinical suspecting of AoD.

CONCLUSION

We highlight that Type A AoD is a life-threatening disease that should be diagnosed immediately at ED. Precise neurological

physical examination should be done immediately for patient with acute LBP. However, the differential diagnoses should not only including neurological disease, but also including vascular disease such as acute AoD. It should be included in the differential diagnosis of patients presenting with LBP and leg numbness, even in a young patient without risk factor and no obvious AoD symptoms. Advocating a lower threshold for use of computed tomography in assessment of unexplained acute LBP with leg weakness should be a practical approach if AoD is highly suspected.

CONFLICT OF INTEREST STATEMENT

The authors have no commercial associations or sourced of support that might pose a conflict of interest.

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